This document was submitted to EPA by a registrant in connection with EPA's evaluation of this chemical, and it is presented here exactly as submitted.

DATA TRANSMITTAL DOCUMENT
Oxamyl
Registration Case Manager – Carmelita White
Registration Product Team Manager - Thomas C. Harris

Name and Address of Submitter

E. I. du Pont de Nemours & Co.
Agricultural Products
Attention: Patricia G. Devine - WM6-121
P. O. Box 80038
Wilmington, DE 19880-0038

Phone: (302) 992-4739

Regulatory Action in Support of Which This Package is Submitted:

Information submitted in support of continued registration of products:

Product Names: DuPont Oxamyl Technical 42® Insecticide/Nematicide, DuPont Vydate® C-LV Insecticide, and DuPont Vydate® L Insecticide/Nematicide

EPA Registration Nos.: 352-400, 352-532, 352-372

Transmittal Date: February 18, 2000

Studies Submitted:

Document No.	Guideline No.	Title	MRID
DuPont Report No. AMR 4391-97, Supplement No. 1	Series 875	Dissipation of Dislodgeable Foliar Residues of Oxamyl from Citrus Following Application of Vydate® L Insecticide in the U.S.A. – Season 1997	
DuPont Report No. AMR 4392-97, Supplement No. 1	Series 875	Dissipation of Dislodgeable Foliar and Soil Residues of Oxamyl Following Application of Vydate® L Insecticide to Tomatoes in the U.S.A. – Season 1997 And 1998	
DuPont Report No. AMR 4393-97, Supplement No. 1	Series 875	Dissipation of Dislodgeable Foliar Residues of Oxamyl from Cucumbers Following Application of Vydate® L Insecticide in the U.S.A. – Season 1997	

Document No.	Guideline No.	Title	MRID
DuPont Report No. AMR 2889-93	164-1	Field Soil Dissipation of Oxamyl Following Application of Vydate® L Insecticide	
DuPont Protocol No. AMR 3143-94	162-4	Degradability and Fate of [1-14C]Oxamyl in Water/Sediment Systems	

Submitter: Patricia G. Devine

Patricia G. Devine
Registration Coordinator

Feb. 18, 2000
Date

Company Name: E.I. duPont de Nemours and Company

Company Contact: Patricia G. Devine

Form Approved OMB No. 2070-0060

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
401 M Street, S.W.
WASHINGTON, D.C. 20460

Paperwork Reduction Act Notice: The public reporting burden for this collection of information is estimated to average 1.25 hours per response for registration and 0.25 hours per response for reregistration and special review activities, including time for reading the instructions and completing the necessary forms. Send comments regarding burden estimate or any other aspect of this collection of information, including suggestions for reducing the burden to: Director, OPPE Information Management Division (2137), U.S. Environmental Protection Agency, 401 M Street, S.W., Washington, DC 20460.
Do not send the completed form to this address.

Certification with Respect to Citation of Data

Applicant's/Registrant's Name, Address, and Telephone Number	EPA Registration Number/File Symbol
DuPont Company, P.O.Box 800038, Wilmington, DE 19880-0038, Attn: Pat Devine,, 302-992-4739	352-400, 352-532, 352-372
Active Ingredient(s) and/or representative test compound(s) oxamyl	Date February 18, 2000
General Use Pattern(s) (list all those claimed for this product using 40 CFR Part 158) Terrestrial Food Crops	Product Name Dupont Oxamyl Tech 42, Vydate L, Vydate C-LV

NOTE: If your product is a 100% repackaging of another purchased EPA-registered product labeled for all the same uses on your label, you do not need to submit this form. You must submit the Formulator's Exemption Statement (EPA Form 8570-27).

☐ I am responding to a Data-Call-In Notice, and have included with this form a list of companies sent offers of compensation (the Data Matrix form should be used for this purpose).

SECTION I: METHOD OF DATA SUPPORT (Check one method only)

☐ I am using the cite-all method of support, and have included with this form a list of companies sent offers of compensation (the Data Matrix form should be used for this purpose).

☑ I am using the selective method of support (or cite-all option under the selective method), and have included with this form a completed list of data requirements (the Data Matrix form must be used).

SECTION II: GENERAL OFFER TO PAY

[Required if using the cite-all method or when using the cite-all option under the selective method to satisfy one or more data requirements]

☐ I hereby offer and agree to pay compensation, to other persons, with regard to the approval of this application, to the extent required by FIFRA.

SECTION III: CERTIFICATION

I certify that this application for registration, this form for reregistration, or this Data-Call-In response is supported by all data submitted or cited in the application for registration, the form for reregistration, or the Data-Call-In response. In addition, if the cite-all option or cite-all option under the selective method is indicated in Section I, this application is supported by all data in the Agency's files that (1) concern the properties or effects of this product or an identical or substantially similar product, or one or more of the ingredients in this product; and (2) is a type of data that would be required to be submitted under the data requirements in effect on the date of approval of this application if the application sought the initial registration of a product of identical or similar composition and uses .

I certify that for each exclusive use study cited in support of this registration or reregistration, that I am the original data submitter or that I have obtained the written permission of the original data submitter to cite that study.

I certify that for each study cited in support of this registration or reregistration that is not an exclusive use study, either: (a) I am the original data submitter; (b) I have obtained the permission of the original data submitter to use the study in support of this application; (c) all periods of eligibility for compensation have expired for the study; (d) the study is in the public literature; or (e) I have notified in writing the company that submitted the study and have offered (i) to pay compensation to the extent required by sections 3(c)(1)(F) and/or 3(c)(2)(B) of FIFRA; and (ii) to commence negotiations to determine the amount and terms of compensation, if any, to be paid for the use of the study.

I certify that in all instances where an offer of compensation is required, copies of all offers to pay compensation and evidence of their delivery in accordance with sections 3(c)(1)(F) and/or 3(c)(2)(B) of FIFRA are available and will be submitted to the Agency upon request. Should I fail to produce such evidence to the Agency upon request, I understand that the Agency may initiate action to deny, cancel or suspend the registration of my product in conformity with FIFRA.

I certify that the statements I have made on this form and all attachments to it are true, accurate, and complete. I acknowledge that any knowingly false or misleading statement may be punishable by fine or imprisonment or both under applicable law.

Signature	Date	Typed or Printed Name and Title
Patricia G Devine	2/18/00	Patricia G. Devine, Registration Coordincator

EPA Form 8570-34 (9-97) Electronic and Paper versions available. Submit only Paper version.

Form Approved OMB No. 2070-0060

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
401 M Street, S.W.
WASHINGTON, D.C. 20460

Paperwork Reduction Act Notice: The public reporting burden for this collection of information is estimated to average 0.25 hours per response for registration activities and 0.25 hours per response for reregistration and special review activities, including time for reading the instructions and completing the necessary forms. Send comments regarding the burden estimate or any other aspect of this collection of information, including suggestions for reducing the burden to: Director, OPPE Information Management Division (2137), U.S. Environmental Protection Agency, 401 M Street, S.W., Washington, DC 20460. Do not send the form to this address.

DATA MATRIX

Date February 18, 2000	EPA Reg No./File Symbol 352-400, 352-532, 352-372	Page 1 of 2
Applicant's/Registrant's Name & Address DuPont Company, P.O.Box 800038, Wilmington, DE 19880-0038, Attn: Pat Devine,, 302-992-4739	Product Dupont Oxamyl Tech 42, Vydate L, Vydate C-LV	

Ingredient oxamyl

Guideline Reference Number	Guideline Study Name	MRID Number	Submitter	Status	Note
Series 875 (AMR 4391-97,Supp.1)	Foliar Dissipation		352	Own	
Series 875 (AMR 4392-97,Supp.1)	Foliar/Soil Dissipation		352	Own	
Series 875 (AMR 4393-97,Supp.1)	Foliar Dissipation		352	Own	
164-1 (AMR 2889-93)	Field Soil Dissipation		352	Own	
162-4 (AMR 3143-94)	Aquatic Aerobic Metabolism		352	Own	

Signature Patricia G. Devine	Name and Title Patricia G. Devine, Registration Coordincator	Date 2/18/00

Form Approved OMB No. 2070-0060

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
401 M Street, S.W.
WASHINGTON, D.C. 20460

Paperwork Reduction Act Notice: The public reporting burden for this collection of information is estimated to average 0.25 hours per response for registration activities and 0.25 hours per response for reregistration and special review activities, including time for reading the instructions and completing the necessary forms. Send comments regarding the burden estimate or any other aspect of this collection of information, including suggestions for reducing the burden to: Director, OPPE Information Management Division (2137), U.S. Environmental Protection Agency, 401 M Street, S.W., Washington, DC 20460. Do not send the form to this address.

DATA MATRIX

Date February 18, 2000	EPA Reg No./File Symbol 352-400, 352-532, 352-372	Page 2 of 2
Applicant's/Registrant's Name & Address DuPont Company, P.O.Box 800038, Wilmington, DE 19880-0038, Attn: Pat Devine,, 302-992-4739	Product Dupont Oxamyl Tech 42, Vydate L, Vydate C-LV	

Ingredient oxamyl

Guideline Reference Number	Guideline Study Name	MRID Number	Submitter	Status	Note
[redacted]			352	Own	
			352	Own	
			352	Own	
			352	Own	
			352	Own	

Signature Patricia G. Devine	Name and Title Patricia G. Devine, Registration Coordincator	Date 2/18/00

EPA Form 8570-35 (9-97) Electronic and Paper versions available. Submit only Paper version.

Public File Copy

Gross Errors in the Product Chemistry Chapter

(Dated 6/14/99 by K. Dockter)

1. On page 2 under the section “Identification of Active Ingredient” the second statement reads, “Oxamyl ‘melts’ at 100-102° C where it changes to a different crystalline form which melts at 108-110°C.” On page 5 the study citation is MRID 40499702. We agree that is the correct study citation. However, that study states that oxamyl melts at 97-100°C.

Gross Errors in the Residue Chemistry Chapter

(Dated 11/5/1999 by Dr. John S. Punzi)

1. In Table A, page 7 in the apple section under the middle scenario (dilute spray after full bloom), the table is broken into 2 sub-rows. Yet, the information in the two sub-rows appears to be the same. Eliminate the second sub-row for that section.

2. Table A does not show that Vydate CLV is also registered for use on peanuts and tobacco. Only Vydate L is listed for those crops under the "Formulation" column. The rates are correct as listed.

3. Table A, footnote 1, page 25 has two phrases missing. The phrase "all crops except" should be added to the sentence on the PBI for Vydate CLV such that it reads, ""for the 3.77 SC/L [EPA Reg. No. 352-532] a PBI of 4 months has been established for all crops except cantaloupe, carrots, celery, cotton, cucumber, eggplant, honeydew melon, peanuts, pepper (bell and non-bell), potatoes, pumpkin, soybeans, squash, sweet potatoes, tobacco, tomatoes and watermelon." Then add, "For these crops plantback can occur at any time."

4. Table B, page 26. Footnote 7 should also be cited under "Must additional data be submitted" for Eggs and the Fat, Liver, Meat and Meat By-Products of Poultry.

5. Table B, References column. Some of the footnotes noted in the reference column do not seem to align with the information expressed in that footnote. Examples are; footnote 7 for references for 860-1500 for ginger, potatoes, onions, celery, grapefruit, oranges, cottonseed, peanuts, nonbearing fruit, (footnote 7 states that there is no need for animal tolerances); footnote 10 for celery, (footnote 10 refers to garlic and onions); footnote 19 for spearmint, (footnote 19 refers to the need for a cotton gin trash study); and footnote 21 for tobacco, (footnote 21 refers to bananas/plantains).

Gross Errors in the Anticipated Residues and Acute and Chronic Dietary Exposure Chapter

(Dated December 22, 1999 by Dr. John S. Punzi)

1. On page 5, last paragraph states, "Field trial data were used in this assessment and it should be noted that residue data from field trials are for oxamyl **and** oxime." We agree and believe that the statement from page 8 should also be added on page 5 to add clarity, "Since it is presently not possible to estimate the ratio of oxamyl to oxime from these field trials, the residues in/on these commodities were assumed to be entirely oxamyl and consequently were considered to be very conservative."

2. On page 6, second paragraph it states the Agency has used a 24-hour period for consumption in the acute dietary analysis. It has been demonstrated that rats' cholinesterase levels reach normal levels two hours after ingestion of oxamyl. We believe the acute dietary risk calculations need to be viewed in two-hour increments rather than over 24 hours. See additional comment section, comment 1.

3. On page 13, under the Pineapples section it is stated, "BEAD was unable to provide reliable data for % crop treated for imports and 100% was used." On November 22, 1999, we supplied the Agency with a letter detailing the use of oxamyl on imported pineapples. (See Attachment 1) Oxamyl is not registered on pineapples in the vast majority of countries that import pineapples into the United States. Even using a conservative estimate, 2.7% of imported pineapple juice, 0.1% of imported fresh pineapple and 2.2% of imported canned pineapple could have been treated with oxamyl.

4. On page 11 the eggplant residue data are discussed. Both tomato and peppers have PDP and/or FDA monitoring data. The Agency's Memorandum "translation of Monitoring Data" dated March 26, 1999 states that tomato or bell pepper monitoring data should be translated to all other crop group 8 commodities. This has not been done for eggplant.

5. We note that some relevant studies were not cited, and were apparently not considered in preparation of this chapter. We believe each of these studies to be important to conducting realistic estimates of dietary exposure. See additional comment section, comment 2.

Additional Comments on the Anticipated Residues and Acute and Chronic Dietary Exposure Chapter

1. On page 6, second paragraph under the section “Consumption Data and Dietary Risk Analysis”, it states “for acute risk assessments, a food consumption distribution is calculated for each population subgroup of interest based on one day consumption data.” However, in the HIARC chapter they state (page 8), “The ChEI was reversible as determined in the carbamate reversibility study (i.e. no cumulative toxicity; recovery of clinical signs of ChEI and ChEI occurred within 2 hours post dosing of 1 mg/kg oxamyl)”. DuPont believes that for acute dietary risk assessments the risk calculation needs to be viewed in 2-hour increments, as that is the length of time for ChEI to fully reverse after ingestion of oxamyl, as noted in the HIARC chapter.

2. We note the following studies were not cited and were apparently not considered in preparation of this chapter. We believe each of these studies to be important to conducting realistic estimates of dietary exposure.

Carbamate reversibility study – as cited above in comment 1 and in the HIARC document – MRID 44472001.

Carbamate Marketbasket study – A study of residue levels on 400 single serve samples for eight different crops. The Agency has stated that marketbasket survey data is the best data for use in acute dietary risk assessment. Use of this data will dramatically reduce dietary exposure for several oxamyl crops. The third interim report is available (MRID 44985601). The final report will issue in April 2000.

Methomyl Processing studies – These studies were used in the methomyl dietary risk analysis by the Agency. Oxamyl and methomyl are structurally similar. They share many of the same chemical and physical properties. Both have been shown to degrade in the environment, plants and animals in a similar manner and timeframe. Oxamyl is even more water-soluble than methomyl. We believe these processing reduction factors should be applied to oxamyl crops as well.

Apple washing, peeling, cooking – MRID 42810701
Lettuce washing and trimming – MRID 42810702
Citrus washing and peeling – MRID 42896901
Green bean canning – MRID 42896902

Gross Errors for the Pesticide Poisoning Incident Data Chapter

(Dated October 1, 1996 by Dr. Virginia A. Dobozy)

1. On page 2 in the "Incident Data System" section, it is mentioned that "one of the reports involved 4 cows, which died after ingesting oxamyl". It is true that on November 16, 1992 we filed a preliminary report with the Agency as required by FIFRA Section 6(a)(2) stating that we had been advised of the death of four cows where oxamyl was alleged to have played a role. However, on March 26, 1993 we filed supplemental information about this incident based on factual information. (See attachment 2) In that submission we noted that the preliminary information was incorrect and that only two cows had died. The other two cows survived and exhibited signs of toxicity for two days after the exposure, which is not consistent with poisoning by a carbamate, such as oxamyl. The cows were exposed to many other possible toxic agents. We could find no record of sales of Vydate® Insecticide to any farmers in that area. DuPont conducted analyses on the containers found at the scene and some of the liver samples. Oxamyl was not found in either. We also attached the findings of the Idaho State Department of Agriculture investigation, which included a statement from the State's toxicologist saying he could not determine the cause of death of the two cows. In fact, his comments include, "Several of the findings are more indicative of other types of toxicity."

As part of initiating the Incident Data System, the Agency pledged that they would also include supplemental information they received. This has not been done here. The preliminary information alleging that oxamyl played a part in the death of four cows needs to be removed. It is not factual information.

2. Key omissions in this chapter:

A statement about the dramatic decline in the number of oxamyl agricultural incidents in California from 1982 to 1994, as shown in Table 3. This decline reflects the labeling changes and other stewardship measures implemented by DuPont during this time period.

The chapter concludes by making two recommendations, both of which DuPont has already done. The fact that DuPont has previously implemented these steps as part of its product stewardship should be noted in the recommendation section.

DuPont has cancelled all uses on ornamentals to address the concern about accidental ingestion in plant nurseries.

DuPont has wording on our labeling requiring chemigation lines to be posted "STOP – Pesticide in Irrigation Water".

Gross Errors in the FQPA Safety Factor Committee Chapter

(Dated September 13, 1999 by Brenda Tarplee)

1. On page 3 in the second paragraph under the "Dietary (Drinking Water) Exposure Considerations" section it states, "The maximum application rate for oxamyl (12 lb a.i. per acre per year – Pineapple scenario)…."

We note that as of March 18, 1999 the pineapple rate is 8 lbs. a.i. per acre per year. Our highest maximum seasonal rate is yams (PR only), 12 lbs. a.i. per acre per year; ginger (HI only), 10 lbs. a.i. per acre per year; and 9 lbs. a.i. per acre per crop for potatoes grown outside the Northeast & Mid-Atlantic states and California.

Gross Errors in the Report of the Hazard Identification Assessment Review Committee
(Dated August 31, 1999 by Dr. Guruva Reddy)

1. On page 5, the opening sentence states that oxamyl is registered on ornamentals. It is not. This use was cancelled in 1996.

2. On page 5, the second sentence states that risk assessments are needed for residential exposures. There are no residential uses of oxamyl, which is correctly noted later in the report.

3. On page 7, the Committee discusses the selection of the chronic reference dose (RfD). We disagree that an acute NOEL should be used for a chronic endpoint. We believe the NOEL from the chronic dog study to be 1.36 mg/kg/day and the RfD would then be set at 0.0136 mg/kg/day. We also believe that the lack of measurement of cholinesterase at time of peak effect in the chronic studies is irrelevant for setting a chronic reference dose. (See additional comments section, comment 1.)
- We also disagree with the conclusion in this section that the chronic dog NOEL is 0.9 mg/kg/day. (See additional comment section, comment 2.)

4. On page 10, in section 4, "Long Term Dermal" it states that oxamyl is registered on greenhouse food and non-food crops. We are not aware of any current registrations of oxamyl on greenhouse crops. The registration of oxamyl on greenhouse ornamental crops was cancelled in 1996.

5. On page 10, in section 5, "Inhalation Exposure" the endpoint is selected based on a NOEL from the acute oral neurotoxicity study. This decision does not conform to the Agency's guidance document, *The Toxicity Endpoint Selection Process*, J. Rowland, February 1997. (See additional comments section, comment 3.)

6. On page 13 in the "Gene Mutation section there is a typographical error. It states the tests in *S. typhimurium* strains TA1535, TA 1547, etc. "TA 1547" should be TA 1537".

7. On page 14 in the "Chromosomal Aberrations" section, it states, "the test was negative up to cytotoxic concentrations (≤70 µg/mL – S9….)". The study actually showed the test was negative up to cytotoxic concentrations (≤100 µg/mL – S9).

8. On page 14 in the "Other Mutagenic Mechanisms" section for the *in vitro* unscheduled DNA synthesis in primary rat hepatocytes, it states, "the test is negative up to cytotoxic concentrations (≤ 5nM)." It should read, "the test is negative up to cytotoxic concentrations (≤10mM)."

9. Page 20, Section VI, "Hazard Characterization", first paragraph, last sentence, the correct CAS number for oxamyl is 23135-22-0.

10. Page 20, Section VI, "Hazard Characterization", third paragraph, there is a typo. The last word, "oxamy" should be "oxamyl".

Additional Comments on the Report of the Hazard Identification Assessment Review Committee

1. On page 7, the Committee discusses the selection of the chronic reference dose (RfD). We disagree that an acute NOEL should be used for a chronic endpoint. We believe the NOEL from the chronic dog study to be 1.36 mg/kg/day and the RfD would then be set at 0.0136 mg/kg/day.

In both this document and the HED Chapter, EPA notes that the acute neurotoxicity endpoint is lower than that of the subchronic neurotoxicity endpoint as well as those of the chronic rat and dog studies. The Agency notes that in the subchronic and chronic toxicity studies, cholinesterase inhibition was not measured at the time to peak effects, implying that this is the reason for differences in NOEL's. DuPont disagrees with this interpretation and instead believes that the apparent increased sensitivity in the acute neurotoxicity study is attributed to the manner and timing of dose administration.

In the acute oral neurotoxicity study, where oxamyl was delivered as a single bolus gavage dose, the NOEL was 0.1 mg/kg. However, in the subchronic neurotoxicity study where oxamyl was delivered in feed for 90 days, the NOEL was 2.1 mg/kg/day. Because carbamate-induced cholinesterase inhibition is rapidly reversible, a study in which the dose is slowly delivered over an extended period of time is expected to produce less of an effect than a study in which the dose is delivered as a single bolus. The difference is related to the feeding pattern of the rats, such that the dose is ingested over the course of many hours in the dietary study. The rate of reversibility of oxamyl-induced cholinesterase inhibition in rats is quite rapid. In a recovery study, male and female rats given a single gavage dose of 1mg/kg of oxamyl had complete recovery of cholinesterase inhibition within two hours (HLR 1997-00641, MRID 44472001). Thus, rats exposed to small doses delivered in the diet over the course of the day recover between feedings.

DuPont wishes to reiterate the inappropriateness of using the acute neurotoxicity study to set the oxamyl chronic toxicity endpoint. The purpose of a chronic reference dose is to assess the effect from steady and prolonged exposure. The effect from a single dose is evaluated in the acute assessment, which does require measurement of cholinesterase at time to peak effect. It is generally most appropriate that the endpoint chosen for risk assessment is by a similar route of administration and of comparable duration as to the expected human exposure. Thus, absent other over-riding factors, the chronic dietary risk assessment should be based on an endpoint representing chronic exposure. The selection of an acute gavage study is inappropriate because gavage dosing does not assess the impact of concurrent consumption of food. In chronic studies, animals are administered the test substance in their feed *ad libidum*, so time to peak effect is less critical than it is in an acute study where chemical is administered by bolus dose. A valid concern could be raised if chronic exposure led to sustained ChE inhibition, which was then exacerbated by an acute exposure. However, this scenario will not be the case with oxamyl, due to the rapid reversibility of its effects. Therefore, DuPont believes the chronic RfD should be based on the most sensitive species in chronic studies, the dog. We believe the NOEL from the chronic dog study to be 1.36 mg/kg/day and the RfD should then be set at 0.0136 mg/kg/day.

2. We disagree with the conclusion that the chronic dog NOEL in male dogs is 0.9 mg/kg/day. A comment in the HED document states that "At 50 ppm...although marked (20%) brain ChE inhibition may not be statistically significant, but considered biologically relevant, since tremors were observed at 150 and 250 ppm...in a previous study". DuPont disagrees that brain ChE was affected on this study. Although the average was somewhat lower for one brain region (cerebellum) for the 50 ppm group, there was large amount of individual variability in brain ChE activity (within and between groups). ChE activity in the cerebellum of individual dogs ranged from a low value of 389 mu/mL for one individual in the 20 ppm group to a high of 1582 mu/mL for one dog in the 12.5 ppm group. Additionally, there was no

dose-response for cerebellar ChE activity nor was there any correlation with ChE activity of other brain regions or with RBC or plasma ChE. Therefore the difference in brain ChE activity of 811 mu/mL in the 50 ppm group compared to 1016 in controls is considered spurious and not compound related.

Mean ChE in mu/mL

	0 ppm	12.5 ppm	20 ppm	35 ppm	50 ppm
Cerebrum	389	333	427	378	416
Cerebellum	1016	977	661	994	811
Caudate Nucleus	539	1116	461	710	611

3. On page 10, in section 5, "Inhalation Exposure" the endpoint is selected based on a NOEL from the acute oral neurotoxicity study. This decision does not conform to the Agency's guidance document, *The Toxicity Endpoint Selection Process*, J. Rowland, February 1997.

DuPont considers the use of the oxamyl acute oral neurotoxicity endpoint for inhalation exposure to be inappropriate. *The Toxicology Endpoint Selection Process* recommends that "extrapolation from oral exposure to inhalation should generally be avoided unless data are available to document that the absorption and metabolism of the pesticide is essentially the same by both routes of exposure….When a short-term exposure assessment is required and only an acute inhalation study is available, the Committee must rely on this study and a NOEL may have to be extrapolated." Thus, following the Agency's own guidance, it is much more appropriate for the inhalation risk assessment to be based on data from oxamyl acute inhalation studies.

Two acute inhalation studies are available with oxamyl that demonstrate, by comparison to acute oral endpoints, toxicity via inhalation is less severe than by the oral route. In both, LC50s were established and the lowest concentration tested produced no deaths, but did produce clinical signs (LOELs). NOELs were not established in either inhalation study. Nonetheless, the lethal levels and LOELs in these inhalation studies can be used as a point of comparison to acute oral studies. In a 4-hour inhalation study with 95% oxamyl technical (MRID 00066902), the LC50 was calculated to be 0.064 mg/L. A concentration of 0.020 mg/L produced no deaths, but clinical signs of cholinesterase inhibition were present. Similarly, in a 4-hour inhalation study study with 42% oxamyl formulation (MRID 40606504), the LC50 was 0.11 mg/L (equivalent to 0.045 mg/L active ingredient). At the lowest dose tested, 0.055 mg/L formulation (0.023 mg/L a.i.), there were no deaths. There were clinical signs of wet fur and ocular and nasal discharge. These inhalation concentrations can be converted to a mg/kg dose by using the exposure duration and the weight and respiration rate of the test rats as recommended in *Inhalation Risk Assessments and the Combining of Margins of Exposure*, J. E. Whalen and H. M Pettigrew, February 10, 1997. EPA has used this method previously, e.g. in the Oxydemeton Methyl Occupational and Residential Exposure Assessment, August 3, 1998.

The acute oral LD50 is 2.8 mg/kg (MRID 00063011). The LOEL and NOEL are 0.75 mg/kg and 0.1 mg/kg, respectively, as determined by cholinesterase inhibition activity in the acute neurotoxicity study (MRIDs 44254401 and 44420301). Both studies were conducted by the oral gavage route.

	Acute Oral	Acute Inhalation Endpoints			Acute Inhalation Endpoints		
	mg/kg	mg/L	mg/kg[c]	Ratio to	mg/L	mg/kg[c]	Ratio to
LC/LD50	2.8[d]	0.064	11.1	3.9x	0.045	7.8	2.8x
LOEL	0.75[e]	0.020	3.5	4.7x	0.023	4.0	5.3x
NOEL	0.1[e]	ND[f]	--	--	ND[f]	--	--

a Acute Inhalation Study HLR 280-69 (MRID 00066902)

b Acute Inhalation Study HLR 199-88 (MRID 40606504)

c Conversion to mg/kg by the method of Whalen and Pettigrew, EPA, Feb. 10, 1997; mg/kg = endpoint in mg/L x 10.26 L/hr (SD rat respiration rate) x 4 hr exposure/ 0.236 kg (SD rat weight)

d Acute Oral LD50 Study HLR 775-80 (MRID 00063011), male LD50 was 3.1 mg/kg; female was 2.5 mg/kg.

e Acute Neurotoxicity Study HLR 1118-96 and HLR 1118-96 Supplement 1 (MRIDs 44254401 and 44420301)

f ND = Not determined

The LD50s and LOELs determined in inhalation studies can be compared on a mg/kg basis to those in oral gavage studies. In all cases, the inhalation endpoints are less sensitive than the oral endpoints. It is perhaps more appropriate to compare the LD50 values, because in the case of LOELs, the inhalation values are based on clinical signs whereas the oral LOELs are based on cholinesterase inhibition. Even using the LD50 comparison, rats are approximately three times less sensitive by the inhalation route than the oral route.

The lower sensitivity of rats in the inhalation studies is not unexpected and reflects differences in the uptake of oxamyl by these two routes. Because the effects of oxamyl on cholinesterase is rapidly reversible, a study in which the dose is delivered over an extended period of time, such as in the inhalation study, would be expected to produce less effect than a study in which the dose is delivered in a single bolus dose, such as an acute oral study. This in fact has been demonstrated in acute and subchronic studies conducted by the oral route. In the acute oral neurotoxicity study (HLR 1186-96, MRID 44254491), where oxamyl was delivered as a single bolus gavage dose the NOEL was 0.1 mg/kg. However with the subchronic neurotoxicity study (HL-1998-00798, MRID 44504901) where oxamyl was delivered in feed, the NOEL was 2.1mg/kg/day, or over twenty-fold less sensitive, even though the rats were exposed for an extended period of time. The difference is related to the feeding pattern of the rats; in the subchronic study the dose was ingested over the course of many hours. The reversibility of cholinesterase inhibition by oxamyl in rats is rapid, with complete recovery of clinical signs and cholinesterase levels within two hours after dosing (HLR 1997-00641, MRID 44472001).

The differences between the inhalation and oral route can serve as an example of where EPA should avoid extrapolation from oral exposure to inhalation because of differences in effective dose between the two routes. There are several approaches that could be used to derive a more realistic value for inhalation exposure assessment.

A NOEL could be extrapolated from an inhalation dose.

The lowest LOEL was 0.02 mg/L (equivalent to 3.5 mg/kg). Following the example found

in the Oxydemeton Methyl Occupational and Residential Exposure Assessment, August 3, 1998, where a conservative factor of 3x was applied to extrapolate a NOEL, the resultant oxamyl inhalation NOEL would be 1.17 mg/kg. We believe that this approach is the most appropriate.

If an oral study were to be used for the inhalation endpoint, it would be more appropriate to use an oral feeding study such as the subchronic neurotoxicity study.
The NOEL from the subchronic neurotoxicity study is 2.1 mg/kg.

If the acute oral neurotoxicity study is used for the inhalation endpoint, at a minimum, a conversion factor of 3x should be applied. This represents the difference in "sensitivity" (actually in distribution and recovery time) between a bolus oral dose and an inhalation exposure extending over time. If this approach were used the inhalation NOEL would be the equivalent of 0.3 mg/kg (0.1 mg/kg from the acute neurotoxicity study x a factor of three).

Gross Errors in the Occupational Exposure and Risk Assessment

(Dated December 8, 1999 by Renee Sandvig)

1. Page 4, Table 2 – The Agency has ignored their own guidance for establishing the inhalation endpoint. See comment 5 in the Gross Errors in the Report of the Hazard Identification Assessment Review Committee section and see comment 3 in the Additional Comments on the Report of the Hazard Identification Assessment Review Committee section.

2. Page 7, Handler Exposures and Assumptions – The Agency does list our current label Personal Protective Equipment (PPE) correctly. However, later in Table 4, MOE's with additional PPE, the MOE's are calculated without consideration of the use of a chemical-resistant apron and/or headgear. The use of a chemical-resistant apron by mixer/loaders should provide at least as much additional protection (50%) as another layer of clothing. Chemical resistant headgear should provide airblast applicators with some additional level of protection.

3. In the same section, it notes that "calculations of handler scenarios are completed using the maximum application rates on the available oxamyl labels." However, in Table 3 the rate used for aerial application to cucurbits (4 lb/A) is a soil rate. Ground rigs traditionally do soil applications. The highest aerial application rate on our label is 3 pounds of oxamyl per acre applied to mint.

4. Page 8, first paragraph – The Agency has used 1200 acres as the default acreage for aerial treatment of cotton. However, the HED Science Advisory Council for Exposure, Policy #9 document states that 1200 is the "upper range" number of acres that could be treated. We believe 350 acres should be used. (See additional comment section, comment 2)

5. Page 14, Table 5 – The Agency notes in footnote (b) that closed mixing and loading systems provide 98% protection factor. Yet, the inhalation unit exposure numbers found in Table 5 for mixing and loading are not 98% more protective than the baseline case (1.2 ug/lb ai vs 0.083). A different data set from PHED has been used here than in the baseline case (Table 3), resulting in protection factor much lower than 98%. It appears as if PHED closed system data from emulsifiable concentrate formulations have been used. We have not been able to identify the source of the PHED closed system trials, but assume they are products that are volatile, such that the inhalation exposure is 3.5 times higher than when the 98% protection factor is used. Such data would not provide a reliable estimate of inhalation exposure to oxamyl in a closed system because of oxamyl's very low vapor pressure (3.8 X 10-7 mm Hg). In the absence of appropriate surrogate data, the use of a default protection factor (98%) is more appropriate.

6. Page 19, Post Application Exposure - We strongly disagree with the half-lives the Agency has calculated based on the best-fit regression of the dissipation data in our three dislodgeable foliar residue studies. The Agency has chosen to fit the data with a linear regression using log transformed concentration data that assumes first order kinetics over the entire time frame of the studies. We believe the data should be fit to a non-linear equation that will account for the initial rapid degradation. Using a non-linear curve fit the half-life of oxamyl on foliage is 1-3 days and 5 days in soil.

The data from the date of application to the date when the residues approach the LOQ is the most significant data for the purpose of determining a re-entry interval. The long tail of additional data points just above the LOQ is not needed to establish a safe re-entry period. In a linear fit with log transformed data the residues at or close to the LOQ from days 5-28 or 35 are weighted as heavily as the data over the 1-5 day period. As a result the initial rapid decline of oxamyl on foliage, which reduces the residue to 0.1 of the initial value by day 5, is masked by the curve fitting routine. A non-linear fit weights the initial

data points more heavily and gives a better description of the decline in oxamyl residues during the critical period when the residues are at a concentration of concern in the evaluation of worker safety. The non-linear curve fitting approach has been advocated by USEPA EFED for determination of pesticide half-lives in soil when the decline curve clearly do not fit a linear first order curve fit (David Jones, EFED).

Concurrent with this letter we are submitting supplemental reports for our three dislodgeable foliar residue studies where we provide our comments on the Agency's review of the studies. Using our dissipation data result in reentry intervals shorter than currently in the draft chapter.

7. Page 20, Assumptions – We believe the transfer coefficients used as default values are overly conservative. (See additional comment section, comment 3)

Additional Comments on the Occupational Exposure and Risk Assessment

1. The handler exposure section presents MOE calculations that are very conservative based on assumptions that are atypical for oxamyl:

- 1200 acres of cotton treated aerial per day (350 per day is typical)
- Soil applications made aerially. (Soil applications would be made by ground)
- Maximum use rates used for all crops (rare to use max. rate)
- Oxamyl exclusively mixed/loaded/applied for 8 continuous hours (mixing/loading of other products would also be typical during the workday.)
- EC formulation data used for closed system scenario (oxamyl formulations are not EC's, they are soluble liquids and oxamyl's vapor pressure is very low)

In addition, oxamyl data not included in Tables 3, 4 and/or 5:

- Correct inhalation endpoint
- All current label PPE not included in Table 4
- Closed system protection factor of 98% not used in Table 5
- Label already requires flaggers to be in enclosed cabs
- Oxamyl has low vapor pressure so inhalation exposure for mixer/loaders will be lower

Considering the overly conservative assumptions and omissions outlined above and the fact that the number of oxamyl handler incidents is very low in the 1990's suggest that the current label PPE provide an adequate Margin of Exposure for workers.

2. Page 8, first paragraph – The Agency has used 1200 acres as the default acreage for aerial treatment of cotton. However, the HED Science Advisory Council for Exposure, Policy #9 document states that 1200 is the "upper range" number of acres that could be treated. The same document states that 350 acres is typical. It is unlikely that any aerial applicators would mix/load and apply oxamyl exclusively on any given day; and therefore would be unlikely to load the amount of oxamyl necessary to treat 1200 acres of cotton. We believe 350 acres is a much better estimate of amounts of oxamyl to which an aerial handler would be exposed. Also, the Agency has been using 350 acres treated aerially in almost all of the OP insecticide RED's, some of which would be marketplace alternatives to oxamyl. The Agency should be consistent in the assumptions used for these risk assessments.

3. Page 20, Assumptions – We believe the transfer coefficients used as default values are overly conservative. The Agency has recently published a RED for an OP where much lower transfer coefficients were used. The Agency has stated in the past that transfer coefficients are a function of the work activity, not the chemical involved. . In addition, new data from the Agricultural Reentry Task Force indicates that the actual transfer coefficient for harvesting of tree fruit is much lower. Using similar transfer coefficients would result in oxamyl maintaining its present 48-hour reentry interval.

4. We believe the Agency's assessment of safe reentry intervals is overly conservative for the following reasons:

- Wrong regression fit of dissipation data to generate half-lives
- Transfer coefficients are too high
- Very few reentry incidents, no indication whether present REI was violated
- Maximum rates assumed to have been used

We believe that in considering all of the above, our current 48 hour REI provides for acceptable safety to reentering workers.

Gross Errors in the HED Chapter

(Dated December 30, 1999 by Christina Jarvis)

1. On page 1 of the overview, first paragraph, it states that oxamyl is formulated "as a solid/technical (42 percent active ingredient)". Our 42% Technical is a liquid.

2. On page 2, the paragraph just below Figure A state the acute dietary risk range from 31-160% of the aRfD. The Dietary Risk chapter states the upper value is 159%.

3. On page 5, section 3.1 "Hazard Profile", second paragraph states that higher NOELs were seen in the chronic studies versus the acute neurotoxicity study. EPA notes that the acute neurotoxicity endpoint is lower than that of the subchronic neurotoxicity endpoint as well as those of the chronic rat and dog studies. The Agency notes that in the subchronic and chronic toxicity studies, cholinesterase inhibition was not measured at the time to peak effects, implying that this is the reason for differences in endpoints. DuPont disagrees with this interpretation and instead believes that the greater sensitivity in the acute neurotoxicity study has more to do with how the material is administered. (See additional comments section, comment 1.)

4. In Table 1, page 7 in the prenatal developmental in rodents "results" section, after the LOEL is stated the following phrase should be added, "(not a developmental toxicant)" as was done in the prenatal developmental in non-rodents "results" section.

- Table 1, page 7 under a summary of the reproduction and fertility effects study, in the results column, the abbreviation "HOT" is used twice. We believe this should read "HDT".

- Table 1, page 7 under the "results" section for the chronic dog study, the cholinesterase NOEL is listed as 0.9 mg/kg.day. We disagree and believe the NOEL is 1.36 mg/kg/day. See comment 3 in the Gross Errors in the Report of the Hazard Identification Assessment Review Committee section and comment 2 in the Additional Comments on the Report of the Hazard Identification Assessment Review Committee section.

- Table 1, page 8 in the "Gene Mutation section there is a typographical error. It states the tests in *S. typhimurium* strains TA1535, TA 1547, etc. "TA 1547" should be TA 1537".

- Table 1, page 8 in the "Chromosomal Aberrations" section, it states, "negative up to cytotoxic concentrations (70 µg/mL – S9….)". The study actually showed the test was negative up to cytotoxic concentrations (100 µg/mL – S9).

- Table 1, page 8 in the "Other genotoxic tests, Unscheduled DNA synthesis", it states the doses were "up to 5nM". However, the doses were actually up to 10mM."

5. On page 11, the last sentence begins discussion of the chronic RfD. We disagree with the chronic RfD selected. See comment 3 in the Gross Errors in the Report of the Hazard Identification Assessment Review Committee section and see comment 1 in the Additional Comments on the Report of the Hazard Identification Assessment Review Committee section.

6. On page 12, the inhalation endpoint is discussed. We believe the Agency has deviated from its own guidance when setting this endpoint. See comment 5 in the Gross Errors in the Report of the Hazard Identification Assessment Review Committee section comments and see comment 3 in the Additional

Comments on the Report of the Hazard Identification Assessment Review Committee section.

7. Some of the percent PAD numbers in Tables 4 and 5 (p. 18-19) do not agree with similar tables on page 15 and 16 of the Anticipated Residues and Acute and Chronic Dietary Exposure chapter.

Additional Comments on the HED Chapter

1. On page 5, section 3.1 “Hazard Profile”, second paragraph states that higher NOELs were seen in the chronic studies versus the acute neurotoxicity study. EPA notes that the acute neurotoxicity endpoint is lower than that of the subchronic neurotoxicity endpoint as well as those of the chronic rat and dog studies. The Agency notes that in the subchronic and chronic toxicity studies, cholinesterase inhibition was not measured at the time to peak effects, implying that this is the reason for differences in endpoints. DuPont disagrees with this interpretation and instead believes that the greater sensitivity in the acute neurotoxicity study has more to do with how the material is administered.

In the acute oral neurotoxicity study, where oxamyl was delivered as a single bolus gavage dose, the NOEL was 0.1 mg/kg. However, in the subchronic neurotoxicity study where oxamyl was delivered in feed for 90 days, the NOEL was 2.1 mg/kg/day. Because carbamate-induced cholinesterase inhibition is rapidly reversible, a study in which the dose is slowly delivered over an extended period of time is expected to produce less of an effect than a study in which the dose is delivered as a single bolus. The difference is related to the feeding pattern of the rats, such that the dose is ingested over the course of many hours in the dietary study. The rate of reversibility of oxamyl-induced cholinesterase inhibition in rats is quite rapid. In a recovery study, male and female rats given a single gavage dose of 1mg/kg of oxamyl had complete recovery of cholinesterase inhibition within two hours (HLR 1997-00641, MRID 44472001). Thus, rats exposed to small doses delivered in the diet over the course of the day recover between feedings.

Gross Errors in the Tier II Estimated Environmental Concentrations Chapter

(Dated October 28, 1999 by Dr. E. Laurence Libelo)

1. Page 5, Table 2

- The units for molecular weight, solubility, and Henry's Law constant should be added to the table.

- References for MW, sol., VP, and Henry's should be given.

- The table states a solubility value of 280,000 ppm. Perhaps it is because it is in scientific notation rounded to 1 decimal place, but the value should be 282,000 ppm.

- The Henry's Law constant is incorrect. It should be 3.9×10^{-13} atm m^3 mol^{-1}. See Attachment 3.

- It is assumed that oxamyl is stable to microbial processes in the water column because no aquatic metabolism study was submitted. (See additional comment section, comment 1)

- The maximum application per year for cotton is incorrectly listed as 1 lb ai/acre. It should be 4 lb ai/A. The modeling was done with the correct seasonal rate.

2. Page 6, References

- Two documents (USDA, NASS, Ag-Census and US Dept. of Commerce 1994a) were cited, but were not included in the reference section.

Additional Comments on the Tier II Estimated Environmental Concentrations Chapter

1. Page 5, Table 2 - It is assumed that oxamyl is stable to microbial processes in the water column because no aquatic metabolism study was submitted. This is mentioned several times in the EFED chapter as well. In the absence of aquatic metabolism data the current EFED guidance is to take the aerobic soil metabolism half-life and double it (resulting in a half-life of 40 days). However, for oxamyl modeling it is assumed hydrolysis at neutral pH is the dominant dissipation route for oxamyl. The hydrolysis half-life at pH 7 is 8 days. We have a water sediment study, AMR 3143-94 (submitted concurrently), which reported non-linear DT50's for oxamyl in two systems of about one day. Treating the water sediment data in a linear manner produces half-lives of about 3 days (throwing out one outlier for one of the test systems). Even correcting the water sediment data for hydrolysis losses (the water was pH 7.3-7.5 in both systems) still gives half-lives of about 7 days. Clearly oxamyl is susceptible to transformation losses in the dark due to other processes than hydrolysis.

If the water sediment half-life of ~7 days (instead of assuming "stable" as EFED did) was used in the surface water modeling in conjunction with the hydrolysis rate of 8 days at pH 7, the Tier II surface water EEC's would be lowered. If so, we compare more favorably with restricted use and endangered species acute freshwater/estuarine/marine organism LOC's .

Gross Errors in the Environmental Fate and Effects Division Chapter

(Dated November 9, 1999 by N. E. Federoff, E. L. Libelo and N. Thurman)

1. Page 2, 3rd word at top of page

- Typo, “oxaime” should be “oxime”.

2. Page 2, "Aquatic Metabolism" paragraph

- Aquatic metabolism data is cited as a gap, but state such studies aren't required. In the absence of data it is assumed aquatic metabolism is not a major transformation pathway. Hydrolysis at neutral to alkaline pH and aqueous photolysis at low pH is rapid. We believe a shorter half-life should be used in modeling. (See additional comment section, comment 1)

3. Page 3, Table 2

- References should be added for solubility, VP, Henry's, and Kow.

- A solubility value of 280,000 ppm is used. Perhaps it is because it is in scientific notation rounded to 1 decimal place, but the value should be 282,000 ppm. The temperature at which the solubility was determined is listed as 20 C when it should be 25 C.

- The Henry's Law constant is incorrect. It should be 3.9×10^{-13} atm m^3 mol^{-1}. See attachment 3.

4. Page 5, Table 3

- The MS field soil dissipation study, AMR 2889-93, should be considered here and added as an additional line entry. (Submitted concurrently)

5. Page 7, "Drinking Water Exposure Assessment", 2nd paragraph

- It is unclear how the Agency arrived at a chronic value of 3 ppb for the oxime. Based on the NC ground water study the highest value is 1.2 ppb.

6. Page 8, 1st paragraph

- A Figure 1 is cited, but no Figure 1 can be found in the document.

7. Page 9, last paragraph

- In the 4th line the units ug/L should be added after "0.018".

- According to our records of the STORET data the 10 detections of oxamyl were from a single station in Lynden, WA, not Whatcom County, CA. We ask for confirmation.

8. Page 13, 3rd paragraph

- The inclusion of the statement that oxamyl might be responsible for honeybee kills with no supporting evidence seems speculative. It should either be supported by factual evidence or removed.

9. Page 16, Table 6

- For the cotton, aerial, multiplication tall grass scenario the chronic RQ should be 7.5 not 6.9.

- The column headings in Tables 5 and 6 should be consistent.

10. Page 18, Table 7

- The equation given in footnote 2 should specify "% body weight consumed as a decimal"

- LOC values should be included at the table base as in Tables 5 and 6.

11. Page 19, Table 8

- In the footnote equation for LD50/ft2 the "/1000" should be removed. It is not used or needed.

- LOC values should be included at the table base as in Tables 5 and 6.

12. Page 19, Table 9

- The avian RQ for tomato should be 4.5, not 5.4.

- On page 52 are the details of how the EEC's and RQ's are calculated for this table. The example calculations are poorly documented. Recalculating the examples as written come up with wrong answers. See comment 18 in this section for more detail.

- For the tomato scenario they cite a 2-lb application rate applied as a sidedress shank. We believe the shank application for tomato should be considered incorporated. EFED assumes it is unincorporated meaning that it is assumed that 100% of the oxamyl is exposed rather than 1%. Also, the Vydate L label specifies that the single application maximum for shank applications to tomatoes in CA is 5 pints of Vydate L/A which is 1.25 lb ai/A, not 2 lb ai/A.

- LOC values should be included at the table base as in Tables 5 and 6.

13. Pages 21-24, Tables 10, 11, 12, 13

- Sufficient details on the modeling parameters/weather sets are not provided to allow us to readily duplicate the reported PRZM/EXAMS EEC's.

14. Page 29-31, "References with no MRID number" section

- Some references like Willis and McDowell, 1987 were cited in the text, but not included in the references.

15. Page 32, Appendix A

- The current/correct/acceptable chemical names for the structures EFED give in Appendix A are:

D1410 (oxamyl)
IUPAC: *N*,*N*-Dimethyl-2-methylcarbamoyloximino-2-(methylthio)acetamide
CAS: Methyl 2-(dimethylamino)-*N*-[[(methylamino)carbonyl]oxy]-2-

oxoethanimidothioate
CAS No.: 23135-22-0

A2213 (oxamyl oxime)
IUPAC: 2-Hydroxyimino-*N*,*N*-dimethyl-2-(methylthio)acetamide
CAS: Methyl 2-(Dimethylamino)-*N*-hydroxy-2-oxoethanimidothioate
CAS No.: 66344-33-0

D2708 (DMOA)
IUPAC: *N*,*N*-Dimethyl-oxalamic acid
CAS: (Dimethylamino)oxoacetic acid
CAS No.: 32833-96-8

16. Appendix C, page 38

- References should be added for solubility, Vapor pressure, and Henry's Law constant.

- A water solubility value of 280,000 ppm is used. Perhaps it is because it is in scientific notation rounded to 1 decimal place, but the value should be 282,000 ppm. The temperature at which the solubility was determined as 20 C when it should be 25 C.

- The Henry's Law constant is incorrect. It should be 3.9×10^{-13} atm m^3 mol^{-1}. (See Attachment 3)

- The maximum application per year for cotton is incorrectly listed as 1 lb ai/acre. It should be 4 lb ai/A. The modeling was done with the correct seasonal rate.

17. Page 46, GENEEC, SCI-GROW

- According to the GENEEC and SCI-GROW input files for pineapple 2 pounds were applied 6 times for 12 pounds total. Our label states the maximum single application rate is 4 pounds and the maximum seasonal rate is 8 pounds per year.

18. Page 52, Terrestrial EEC calculations

- As stated for the page 19, Table 9 comments the calculations don't seem to be accurate.

Listed below is an exact copy of the example given for tomato, which assumes 60-inch row spacing, 2-inch band, and 2 lb ai/A unincorporated. Similar examples are given for potato and carrot.

"0.0002295 lb ai/1,000 ft of row = 2 lb ai/acre/[43,560 sq ft/acre/(60 in row spacing X ft/12 in)]
0.167 Bandwidth (ft) = 2 in x 1 ft/12in
17.38 ai (mg)/sq ft = 453,590 mg/lb x [0.0002295 lb ai/1,000 ft row/(1,000 ft x 0.167 bandwidth (ft))]
17.38 Exposed ai (mg)/sq ft = 17.38 ai (mg)/sq ft x 1 (100 percent unincorporated)

Duck: LD50s/sq ft = 17.38 Exposed ai (mg)/sq ft/3.2 LD50 x 1.2 weight of bird (kgs) = 5.4
Rat: LD50s/sq ft = 17.38 Exposed ai (mg)/sq ft/2.5 LD50 x 0.3 weight of bird (kgs) = 23.2"

Looking at it line by line;

"0.0002295 lb ai/1,000 ft of row = 2 lb ai/acre/[43,560 sq ft/acre/(60 in row spacing X ft/12 in)]"

What is actually calculated is lb ai/ft of row, not per 1,000 ft of row.

"0.167 Bandwidth (ft) = 2 in x 1 ft/12in"

This calculation is correct.

"17.38 ai (mg)/sq ft = 453,590 mg/lb x [0.0002295 lb ai/1,000 ft row/(1,000 ft x 0.167 bandwidth (ft))]"

If you do the calculation as they specify the answer is 0.0006233 mg ai/cubic foot. The number and units make no sense.

If you do the calculation and multiply by "(1,000 ft x 0.167 bandwidth (ft))" rather than divide then the answer matches what they report, 17.38. However, the units become mg ai/ft not mg ai/sq ft.

"17.38 Exposed ai (mg)/sq ft = 17.38 ai (mg)/sq ft x 1 (100 percent unincorporated)"

This calculation is correct.

"Duck: LD50s/sq ft = 17.38 Exposed ai (mg)/sq ft/3.2 LD50 x 1.2 weight of bird (kgs) = 5.4"

The answer should be 4.5 and not 5.4. This should be corrected in Table 9 also. "LD50s" should just be LD50, or explained if it has a different meaning than LD50.

"Rat: LD50s/sq ft = 17.38 Exposed ai (mg)/sq ft/2.5 LD50 x 0.3 weight of bird (kgs) = 23.2"

This calculation is correct. "LD50s" should just be LD50, or explained if it has a different meaning than LD50.

Additional Comments on the Environmental Fate and Effects Division Chapter

1. Page 2, "Aquatic Metabolism" paragraph

- Aquatic metabolism data is cited as a gap, but state such studies aren't required. In the absence of data it is assumed aquatic metabolism is not a major transformation pathway. Hydrolysis at neutral to alkaline pH and aqueous photolysis at low pH is rapid.
The hydrolysis half-life for oxamyl at pH 7 is 8 days, and is a few hours at pH 9. The aqueous photolysis half-life at pH 5 = 7 days. The water/sediment half-life (oxamyl was only in the water column) corrected for hydrolysis losses was ~7 days. A method that may be used to estimate contributions from separate and well-documented loss pathways is the following:

$$\text{half-life}_{\text{modeled}} = \frac{1}{(1/hl_1 + 1/hl_2)}$$

where: $\text{half-life}_{\text{modeled}}$ = half-life value used in the fate modeling
hl_1 = half-life for process 1 (hydrolysis in the water column for example)
hl_2 = half-life for process 2 (microbial transformation in the water column for example)

In the case of oxamyl with an aquatic metabolism half-life of 7 days and hydrolysis half-life of 8 days, the half-life for use in the modeling would be = 3.7 days. From a modeling perspective 3.7 days is significantly shorter than 8 days assumed for just hydrolysis.

2. Page 3, Table 2
- We are concurrently submitting AMR 2889-93, a field dissipation study in MS. Oxamyl dissipated quickly (half-life = 10 days) and was not observed beyond 45 cm depth. This study reinforces that oxamyl dissipates quickly in a wider variety of settings.

THE HENRY'S LAW CONSTANT FOR OXAMYL

The measured vapor pressure and aqueous solubility at 25°C were used to calculate the Henry's Law Constant for oxamyl.

The vapor pressure of oxamyl at 25°C is 3.84×10^{-7} mm Hg (AMR-1267-88) which is converted to 5.05×10^{-10} atmospheres by multiplying by the conversion factor of 1 atmosphere/760 mm Hg.

The aqueous solubility at 25°C is 282 g/liter and the molecular weight is 219.3 g/mole. The solubility of oxamyl, therefore, can be converted to 1.29 moles/liter by dividing the above value by the molecular weight. Using the conversion factor of $1000/m^3$, the solubility can be expressed as 1290 moles/m^3.

Since the Henry's Law Constant is the ratio of the vapor pressure to the aqueous solubility at the same temperature and for the same physical state of the compound, we calculated the Henry's Law Constant of oxamyl at 25°C to be 5.05×10^{-10} atmospheres/1290 moles-m^{-3} or 3.92×10^{-13} atmospheres-m^3/mole.

This value of the Henry's Law Constant indicates the oxamyl has negligible escaping tendency from a dilute aqueous solution. According to Lyman et al, if the Henry's Law Constant is less than about 3×10^{-7} atmospheres-m^3/mole, as it is for oxamyl, the substance is less volatile than water and could be considered essentially nonvolatile(1).

(1). W. J. Lyman, W. F. Reehl, and D. H. Rosenblatt, "Handbook of Chemical Property Estimation Methods", McGraw-Hill, Inc., 1982, p 15-15.